TOXIC ALGAL BLOOMS:POTENTIAL HAZARDS TO SCALLOP CULTURE AND FISHERIES

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Phycotoxins from algal blooms are accumulated by filter-feeding bivalve molluscs. Since only the adductor muscle of scallops has been traditionally marketed, scallops are not usually included in routine monitoring programs but intensified aquaculture ventures in areas prone to toxic blooms have provoked public health concerns. Our focus on the sequestering and biotransformation of phycotoxins in scallops indicate that: 1) toxins are not distributed evenly; toxin is usually concentrated in the mantle and digestive gland; 2) some scallop tissues, e.g. digestive glands and mantles remain highly toxic throughout the year; 3) toxicity varies (43.5%) between individuals in the same area; 4) no correlations could be made between toxicity levels in gonadal and other tissues.

Scallop culture and commercial fisheries can thrive in areas prone to toxic algal blooms if only the adductor muscle is utilized. Safe marketing of "roe-on" scallops is feasible only under strict regulatory regimes. Marketing of mantles or whole scallops poses a high risk to public health and should only be undertaken after extensive monitoring. Scallop mariculturists should be aware of risks associated with phycotoxins. Further, public health guidelines with particular emphasis on toxin levels in individual tissues is necessary if scallops are to be marketed whole or in conjunction with tissues other than adductor muscles.

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The impact of toxic algal blooms on scallop culture and fisheries is often underestimated or even ignored since traditionally only the large adductor muscle is consumed. Adductor muscle tissue is usually free of accumulated toxins of algal origin (phycotoxins), although levels in excess of the regulatory limit may occur. Scallop species are, however, by no means exempt from the effects of toxic algal blooms (Table 1). Generally, scallops are not included in routine monitoring programs for paralytic shellfish toxins and they have only recently been covered by regulations of the Interstate Shellfish Sanitation Conference (ISSC) in the US. Areas prone to outbreaks of toxic algae overlap with areas where scallops are fished or cultured commercially (Fig.1). With expansion of scallop culture and increased interest in marketing non-traditional scallop tissues, as well as whole and 'roe-on' scallops, an understanding of the problems and hazards posed by toxic algae to the scallop industry is required.

Scallops are common inhabitants of nearly every coastal region worldwide and support major commercial fisheries and mariculture industries (Hardy,1991; Shumway,1991; Fig.1). Blooms of toxic and noxious algae are also regular cosmopolitan events (LoCicero,1975; Taylor & Seliger,1979; Anderson et al.,1985; Okaichi et al.,1989; Granéli et al.,1990; Hallegraeff,1993). Their impact on utilization of shellfish resources was reviewed by Shumway (1989, 1990) and Hallegraff (1993). Filter-feeding bivalves, such as scallops, accumulate toxic algae and associated toxins in their tissues rendering them vectors of various types of seafood poisoning, including paralytic shellfish poisoning (PSP), diarrhetic shellfish poisoning (DSP), amnesic shellfish poisoning (ASP). Such shellfish are unfit for human consumption.

While some groups of toxic algae have had devastating effects on scallop populations (Table 1), the primary threat to industry and public health is the potential for human illnesses such as DSP and PSP. Scallop culture and fisheries have been conducted in areas prone to blooms of highly toxic algae, e.g. Canada, Japan, United States, France (Shumway,1990,1991; Fig.1).

Since only the adductor muscle is generally consumed in North America, scallops are usually

FIG.1. Geographical distribution of PSP, DSP, NSP and ASP toxins and their relationship to commercially utilised (fished and cultured) scallop genera.

shucked at sea where shells and unwanted tissues are discarded. These 'other' tissues include the mantle (rims or rings), gonad (roe), digestive gland (hepatopancreas, liver), and gills; together they comprise over 80% of the total weight of tissue (Schick et al.,1992; Fig.2). In some areas, e.g. Europe, Japan and southern Australia, scallops are sold with the gonad attached ('roe-on') and there has been a steady and continuing interest in fuller utilization of scallop tissues in other regions, particularly the US and Canada (Bourne & Read,1965; Dewar et al.,1971). The idea that consumption of scallops is always safe should not be accepted unreservedly. While scallops are not the most common vectors of paralytic or diarrhetic shellfish poisonings, there have been several reported illnesses and even some deaths attributed to toxic scallops.

Prior to the occurrence of PSP toxins on Georges Banks, Bourne & Read (1965) advocated the marketing of scallop muscles with attached roes and rims (mantles). Dewar et al. (1971) presented procedures and recipes for production of high-quality frozen and canned products and, based on results of Japanese taste panels, indicated consumer acceptance of these products. The sea scallop, *Placopecten magellanicus*, and Japanese scallop, *Patinopecten yessoensis*, support the two largest scallop fisheries worldwide; *P. magellanicus* is the focus of efforts to market roe-on product and whole animals, as is done with *Pt. yessoensis*. There is a renewed interest in marketing both whole and 'roe-on' scallops (*P. magellanicus*) from Canada and the northeastern US (Gillis et al.,1991; Merrill,1992), and whole pink scallops (*Chlamys rubida*) from the Pacific northwest (Nishitani & Chew,1988). However, the first reported incidence of PSP toxins on Georges Banks (Sharifzadeh et al.,1991; White et al.,1992a) and the persistent presence of these toxins in the Pacific northwest (Nishitani &

Chew,1988; Shumway,1990) has sparked new concerns with regard to consumer safety.

Scallops are opportunistic filter feeders which utilize both pelagic and benthic microorganisms as food sources (Shumway et al.,1987; Bricelj & Shumway,1991). These organisms are consumed and concentrated in the digestive gland. Where toxic algal species are present, the shellfish become vectors of shellfish poisons including PSP and DSP. Poisonings due to PSP have been reported after consumption of both sea scallops (Medcof et al.,1947; Washington Office of Public Health Laboratories and Epidemiology,1978), and pink and spiny scallops consumed whole (Canadian Department of Fisheries and Oceans,1989). Seafood poisoning attributed to DSP following consumption of scallops has been known in Japan since 1977, and has resulted in several hundred illnesses (Nomata, pers. comm.). On September 23, 1983, a 5 year old boy died of PSP after eating scallops from Olotayan Island in the Philippines (Estudillo & Gonzales,1984). One death was reported from consumption of *Chlamys nipponensis* in Iwate prefecture, Japan in 1962 (Nomata pers. comm.) and a death was also attributed to consumption of *Hinnites* in California (Sharpe,1981).

Accumulation of PSP and DSP toxins has already had devastating effects on the scallop industry (both cultured and fished), especially in areas such as the Atlantic and Pacific coasts of North America and in Japan where toxic blooms are regular events (Ogata et al.,1982; Gillis et al., 1991; Nishihama,1980). Japan has stopped supplying whole scallops to large markets such as France because of the presence of PSP toxins (Merrill,1992). Careful monitoring of 'roe-on' scallops in Canadian waters resulted in closure of 'roe-on' fishing for most of the Canadian sector of Georges Bank during 1989 and 1990, when PSP toxin levels exceeded the tolerance limit (80μg STXeq/100g) (Gillis et al.,1991). Efforts are currently underway by the National Marine Fisheries Service (NMFS) to develop a protocol for certification of 'roe-on' or whole scallops (*Placopecten* or *Argopecten*) harvested in US federal waters west of 71°W longitude.

Problems associated with scallop toxicity monitoring are exacerbated by high variability in toxicity between individual animals (Whitefleet-Smith et al.,1985; Gillis et al.,1991; White et al.,1992b). This variability can be considerable (Beitler,1991; White et al.,1992b and references therein) and has been attributed to differences in season, geographical location, specific toxins involved and toxin concentrations. Finally, bioconversion of toxins between/within scallop tissues may also account for some of the variation in toxicity.

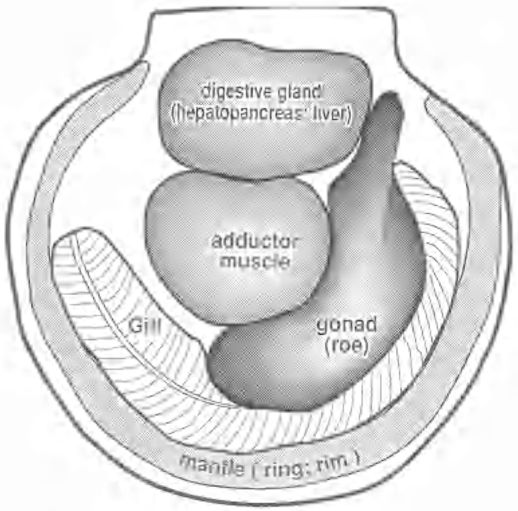

FIG.2. Diagrammatic representation of scallop tissues.

Total toxicity varies not only between locations and individuals, but also among tissues of individual scallops. Most available data on PSP toxin distribution among scallop tissues are for the sea scallop, *P. magellanicus* and Japanese scallop, *Pt. yessoensis* (Table 2). The digestive gland (hepatopancreas, liver) is usually the most toxic tissue; with levels in excess of 45,000μg STXeq/100g, as determined by AOAC mouse bioassay, having been recorded in the Gulf of Maine (Watson-Wright et al.,1989). This is of particular importance for marketing of whole scallops and special care must be taken in any area where toxic algae are present to ensure regular testing for these toxins.

High levels of PSP toxicity have also been reported for gonadal tissue (roe). Watson-Wright et al. (1989) reported detectable PSP toxicity (i.e. 40μg STXeq/100g) in 69% of scallop gonadal samples analyzed (n=41) from the Bay of Fundy, but found no correlation between the toxicity of gonadal tissue and other tissues. While these high toxicity levels (e.g 1300μgSTXeq/100g tissue in *P. magellanicus* from Mascarene, New Brunswick; Microbiology Division, Food Research Laboratories-Health and Welfare, Canada; Black's Harbour, New Brunswick data reports), seem to be the exception rather than the rule, they again point to the need for strict monitoring prac-

TABLE 1. A summary of toxic and noxious algal blooms associated with scallops.

Algal species	Shellfish species affected	Notes	Location	Reference
Dinophysis acuminata D. fortii	Chlamys nipponensis Patinopecten yessoensis Pecten albicans	toxic	Japan	Anraku (1984)
Alexandrium tamarense	Chlamys opercularis *Pecten maximus*	highly toxic; not adversely affected	Northumberland U. K.	Ayres & Cullum (1978);Ingham et al. (1968); Wood & Mason (1968)
A. tamarense	Placopecten *magellanicus*	highly toxic	Gulf of Maine and eastern Canada;Bay of Fundy and St.Lawrence regions	Prakash(1963); Bourne (1965); Caddy & Chandler (1968); Prakash et al. (1971); Medcof(1972); Hurst (1975); Hartwell (1975); Hsu et al. (1978); Tufts (1979); Jamieson & Chandler (1983); Shumway et al. (1988); Gillis et al. (1991); Cembella & Shumway(1991)
A. tamarense	Placopecten magellanicus	highly toxic	Georges Bank, Gulf of Maine	White et al. (1992a,b)
A. tamarense	Chlamys nipponensis Patinopecten yessoensis	toxic	Japan	Oshima et al.(1982)
A. tamarense	Argopecten irradians	toxic	Massachusetts	Bicknell & Collins (1973)
A. tamarense	Patinopecten yessoensis	toxic	Japan	Sekiguchi et al. (1989)
A. tamarense	Placopecten *magellanicus*	violent swimming activity; production of mucus	laboratory	Shumway & Cucci (1987); Gainey & Shumway (1988a,b)
A. tamarense	Pecten maximus	toxic	laboratory	Lassus et al. (1989)
A. tamarense	Patinopecten yessoensis	toxic	Japan	Maruyama et al. (1983)
A. tamarense	C. nipponensis C. nobilis	toxic	Japan	Anraku (1984); Oshima et al.(1982)
Alexandrium	Patinopecten yessoensis	toxic	Japan	Nishihama (1980)
A. catenella	*Patinopecten yessoensis C. nipponensis akazara*	toxic	Japan	Noguchi et al. (1978, 1980a,b, 1984)
A. catenella	*Hinnites multirugosus Chlamys hastata*	toxic; human illness	British Coumhia	DFO (1987; 1989)
A. catenella	*Hinnites multirugosus*	1 death from eating viscera	California	Sharpe (1981)
A. catenella	*Hinnites multirugosus Chlamys hastata,Pecten caurinus,Pecten* sp.	toxic	Pacific USA	Nishitani & Chew (1988)
A. catenella	*Chlamys patagonicus*	toxic	Chile	Avaria (1979);Guzman & Campodonico (1978)
Gymnodinium catenatum	*Equichlamys bifrons, Mimachlamys asperrimus, Pecten fumata*	toxic	Tasmania	Hallegraeff & Summer (1986); Hallegraeff et al. (1989); Oshima et al. (1982, 1987a,b)
G. catenatum	Pecten albicans	first report of toxicity by this species	Japan	Ikeda et al. (1989)
G. breve	*Argopecten irradians*	scallop deaths; recruitment failure	North Carolina	Barris (1988); Tester & Fowler (1990); Summerson & Peterson (1990)

TABLE 1 *(continued)*

Algal species	Shellfish species affected	Notes	Location	References
G. veneficum	*Pecten maximus*	scallop mortality	laboratory	Abbott & Ballantine (1957)
Gyradinium aureolum	*Pecten maximus*	mortalities in young scallops	France	Lassus & Berthome (1988)
Gy. aureolum	*Pecten maximus*	numbers of larvae declining during bloom	Lough Hyne, Ireland	Minchin (1984)
Gy. cf. *aureolum*	*Pecten maximus*	high mortality in post-larvae and juveniles; reproduction and growth inhibited in adults	France	Erard-Le Denn et al. (1990)
Cerotium tripos	*Placopecten magellanicus*	nontoxic; mortalities due to O_2 depletion	New York Bight	Mahoney & Steimle (1979)
Aureococcus anophagefferens	*Argopecten irradians*	larval shell growth reduced and mortalities increased	laboratory	Gallagher et al. (1988)
A. anophagefferens	*Argopecten irradians*	mass mortalities	Long Is, NY; Narragansett Bay, RI; Barnegat Bay, NJ	Cosper et al. (1987); Tracey et al. (1988); Tracey (1985); Smayda & Fofonof (1989)
A. anophagefferens	*Argopecten irradians*	76% reduction in adductor weights; recruitment failure of year class	Long Island, NY	Bricelj et al. (1987)
Rhizosolenia chunii	*Pecten alba*	bitter taste rendered shellfish unmarketable for 7 months; digestive gland lesions and shellfish mortalities	Australia	Parry et al. (1989)
not specified, probably *Alexandrium*	*Patino. yessoensis, Chlomys farreri*	toxic	Korea	Jeon et al. (1988)
not specified	*Chlamys nobilis*	toxic	Japan	Nagashima et al. (1988)

Alexandrium tamarense (=*Gonyaulax tamarensis* =*Protogonyaulax tamarensis*); *A. catenella* (=*Gonyaulax catenella* =*Protogonyaulax catenella*); *Gymnodinium breve* (=*Ptychodiscus brevis*)

tices if scallop products other than adductor muscles are to be utilized.

ADDUCTOR MUSCLE TOXICITY

It had been generally accepted that scallop adductor muscle tissue tends to remain free of accumulated toxins (Medcof et al.,1947; Watson-Wright et al.,1989; Shumway,1990); however, reports of adductor muscles with measurable toxicity, and even scores exceeding the regulatory limit of 80μg STXeq/100g tissue have been reported for several scallop species (Table 1). It is impossible to estimate the toxicity of adductor muscles of scallops based on the toxicity of surrounding visceral tissue (Beitler,1991; Watson-Wright et al.,1989) and no assumptions regarding the toxicity of individual scallop tissues should be made on any such correlations.

DETOXIFICATION

In addition to individual variations in toxin levels and among tissues within individuals, scallops exhibit slow and markedly variable rates of detoxification. All data available are for PSP toxins and are limited to *Pt. yessoensis, P. magellanicus, C. nipponensis akazara* and *Pecten maximus*. Once PSP toxins are accumulated by scallops, they are only slowly eliminated. Detectable PSP toxicity (40μg STXeq/100g) by AOAC mouse bioassay has been reported to persist in *P. magellanicus* for extended periods ranging from several months to two years (Medcof et al.,1947; Jamieson & Chandler,1983; Shumway et al., 1988; unpubl. data). Digestive glands of *Pt. yessoensis* (initial toxicity 34,000μgSTX eq/100g; i.e. 1700MU/g tissue; MU=mouse units) were reported to contain 2,000μgSTX eq/100g (100MU/g) even after being held in running

TABLE 2. Levels of paralytic shellfish toxins (μg STXeq/100g tissue) recorded in scallop species from various geographical locations. A conversion factor of 1 MU =~ 0.20 μg STXeq has been used to standardize data sets.

SPECIES	TISSUE	TOXIN LEVEL (μg STXeq/100g)	LOCATION	REFERENCE
Chlamys nipponensis akazura	Adductor Gonad(ovary) Midgut gland	80 640 4000	Ofunato Bay, Japan	Noguchi et al. (1978)
Chlamys opercularis	Whole	256	Flamborough Head, UK	Ingham et al. (1968)
Chlamys rubida	Adductor	56	Washington, USA	Anonymous (1987)
*Crassadoma gigantea** (=*Hinnites multirogosus*)	Adductor muscle Viscera+ Whole body	130 2500 1200	British Columbia	DFO (1989)
	Adductor muscle Viscera+ Whole body	229 2036 295	Washington, USA	
	Adductor muscle Viscera+ Whole body	2000 26000 13593	California, USA	Anonymous (1980); Sharpe (1981)
Patinopecten caurinus	Adductor	58	Alaska, USA	Anonymous (1987)
Patinopecten yessoensis	Adductor Gonad(ovary) Midgut gland	400 900 16000	Ofunato Bay, Japan	Noguchi et al. (1978)
	Adductor Digestive gland Other	40 2040 220	Funka Bay, Japan	Noguchi et al. (1980a,b)
Patinopecten yessoensis	Digestive gland	20,000	Ofunato Bay, Japan	Sekiguchi et al. (1989)
	Digestive gland	8400	Ofunato Bay, Japan	Kodama et al. (1990)
	Digestive gland	6000	Kawauchi Bay, Japan	Ogata et al. (1982)
	Digestive gland	15000	Funka Bay, Japan	Nishihama (1980)
	Digestive gland Adductor muscle	130,000-220,000 60-260	Japan	Noguchi et al. (1984)
	Hepatopancreas	34,000	Ofunato Bay, Japan	Oshima et al. (1982)
	Digestive gland Rectum Foot Gonad Mantle Gill Adductor muscle	42,000-70,000 4200-12,400 3200-4600 2200-3200 1500-2200 1420-2200 320-860[a]	Ofunato Bay, Japan	Maruyama et al. (1983)
	Digestive gland	15,000	Funka Bay, Japan	Nishihama (1980)
Pecten maximus	Whole	1568	Fame Bank, UK	Ingham et al. (1968)
Pecten maximus	Whole(?)	2700	laboratory	Lassus et al. (1989)
Pecten grandis (=*Placopecten magellanicus*)	Whole Digestive gland Gill Adductor muscle Gonad Other	1520 8000 560 <40 190 680	Lepreau Basin, New Brunswick	Medcof et al. (1947)

TABLE 2. *(continued)*

SPECIES	TISSUE	TOXIN LEVEL (μg STXeq/100g)	LOCATION	REFERENCE
Placopecten magellanicus	Hepatopancreas Gonad	1440 44	Canadian Georges Bank	Gillis et al. (1991)
	Adductor muscle Gonad Hepatopancreas Gill Rims	<40 2400 50,000 570 4500	Bay of Fundy, Canada	Hsu et al. (1979)
Placopecten magellanicus	Adductor Gonad Digestive gland Mantle	<40* 420* 4180* 2830*	Maine, USA	Shumway et al. (1988; unpubl. data)
Placopecten magellanicus	Whole+ Adductor Whole (– adductor)	3888 183* 14775*	Georges Bank (Loran 1336543777)	White et al. (1992a,b); Shumway (unpubl.)
Placapecten magellanicus	Hepatopancreas Gonad Adductor muscle Gills Rims	45,000* 1700* undetectable 250* 4700*	Bay of Fundy, Canada	WatsonWright (1989)
Placopecten magellanicus	Whole Digestive gland Gonad Adductor Gill	2200* 150,000 184–286* 60 100–600	Bay of Fundy, Digby, Canada	Jamieson & Chandler (1983)
	Digestive gland All other tissues	140* <32	N Edge, Georges Bank	Jamieson & Chandler (1983)
	Adductor Digestive gland	120* 25,000	Mascarene, Nova Scotia, Canada	Jamieson & Chandler (1983)
Placopecten magellanicus	Liver[b]	36–66	N Edge, Georges Bank	Bourne (1965)
	Gonad Liver Mantle Adductor Gill	43* 4000* 1440* <32* <32*	Bay of Fundy, Canada	Bourne (1965)

* maximum reported values
\+ whole body minus adductor
[a] probably leached from other tissues; scallops were frozen whole for several months prior to dissection and analysis
[b] stomach and digestive diverticulum

seawater for five months in the laboratory (Oshima et al.,1982).

Cooking can reduce toxin levels considerably (Medcof et al.,1947; McFarren et al.,1960) and canning has been used to reduce toxicity of scallop tissue to acceptable levels (Noguchi et al.,1980a,b); however, as a means of reducing toxicity, canning is usually only effective when toxin levels are relatively low, although Noguchi demonstrated that canning might be applicable for scallops with PSP toxicity at levels as high as 8,000μgSTXeq/100g (400MU/g) tissue.

Freezing does not appreciably reduce toxin levels, although long-term storage at temperatures from 0 to –20°C may lead to some degradation of specific toxins, often to more toxic derivatives. Moreover, freezing of whole scallops can result in migration of toxins from highly toxic

tissues, e.g. digestive gland, into adductor muscle, rendering the latter unsafe for human consumption (Noguchi et al.,1984; unpubl. data). Toxin can also leach from attached gonads to the adductor muscle during shipping (Bruce & Delaney,1972).

PRECAUTIONS

A market for roe-on scallops is feasible only under strict monitoring for algal toxins. Establishment of public health safety guidelines with particular emphasis on toxin levels in individual body parts is a necessity if scallops are to be marketed whole or in conjunction with any tissues other than adductor muscles.

Marketing of rims (mantles) or whole scallops can pose a high risk public health and should only be undertaken under the strictest of monitoring plans. The economic success of such an industry is questionable.

Mariculturists should be acutely aware of the potential risks and dangers associated with toxic algal blooms and the marketing of various scallop products.

Successful culture facilities and commercial fisheries can persist in areas prone to toxic algal blooms; however, only through careful site selection and monitoring can optimal utilization of scallop resources be realized and economic losses kept to a minimum.

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